MYOCARDIAL CONTRACTION IN DIFFERENT PARTS OF THE LEFT VENTRICLE

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The electrical and mechanical activity of different parts of the myocardium shows a number of characteristic variations. This functional heterogeneity of the heart, combined with differences in its architectonics, may be considered to be of great importance for certain adaptive changes in cardiac activity in normal and, in particular, in pathological conditions. It has been shown, for example, that in compensatory hyperfunction of the heart caused by experimental aortic stenosis or mitral incompetence the principal role is played by various parts of the myocardium of the left ventricle [1].

Accordingly, in the present investigation the contractile function of the myocardium in various parts of the left ventricle was studied.

EXPERIMENTAL METHOD

Experiments were carried out on dogs under urethane anesthesia (1.0-1.5 g/kg) in open chest conditions with artificial respiration.

The index of myocardial contractile function used in the experiments was the intramyocardial pressure (IMP), recorded by the method described by Kreuzer and Schoeppe [3]. The principle of the method consists of measurement of the pressure in a needle, filled with physiological saline containing heparin, inserted into the heart muscle. The needle is connected firmly to an electromanometer with very small volume displacement (about 0.01 mm^3/100 mm Hg).

The needle for measuring the IMP (length 25 mm, external diameter 1.2-1.5 mm) has two holes measuring 0.75 mm^2 in its sides, one opposite the other. The end of the needle is closed and pointed resembling a nail. It is fixed with a washer fitted with a screw clip, and sutured to the epicardium after a wide incision in the pericardium. An absolutely rigid connection between the recording needle and the electromanometer proved to be unsuitable for experiments that were at all prolonged, for it led to injury to the contracting heart by the end of the needle, and changes in the configuration and size of the heart influenced the position of the needle in the substance of the myocardium. Accordingly, in the present investigation the needle was connected to the electromanometer, not by a metal tube but by a segment of a standard catheter about 25 cm long.

The anterolateral surface of the left ventricle was divided conventionally into three parts: base, middle third, and apex. In each experiment the IMP was recorded in one of these divisions of the left ventricular wall. The IMP was measured at a depth of 6-12 mm from the outer surface of the heart. The axis of the holes in the recording needle was directed parallel to the interventricular groove. As a rule, the maximal value of the IMP was recorded for each portion of the myocardium.

Concurrently with the IMP, the ECG and the pressure in the left ventricle and aorta were recorded. The recordings were made on a "Galileo" ink-writing instrument (tape winding speed up to 100 mm/sec) and simultaneously on a type N-700 loop oscillograph (winding speed 160 mm/sec).

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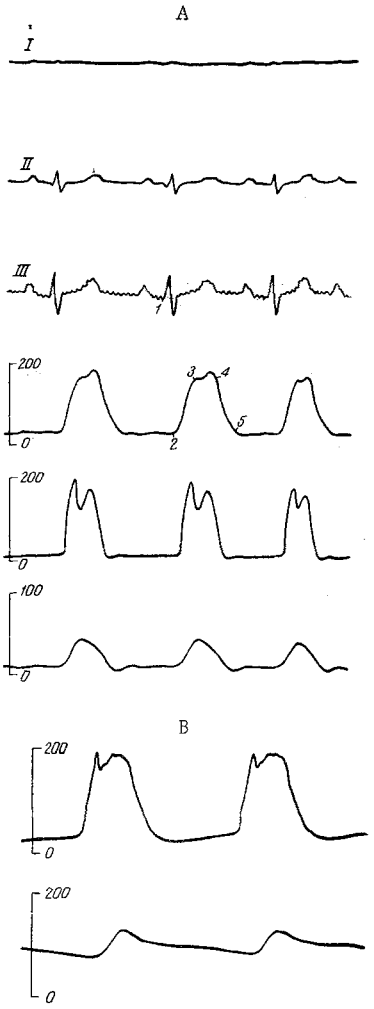

Typical IMP curve for a dog. A (from top to bottom) ECG in leads I, II, and III, IMP, pressure in left and right ventricles; B) bimodal IMP curve and curve of pressure in aorta. Explanation in text.

In 11 experiments the IMP was recorded in the base of the left ventricle, in 6—in the middle third, and in 9—in the apex of the left ventricle.

EXPERIMENTAL RESULTS AND DISCUSSION

The nomenclature of the various phases of the cardiac cycle proposed by Kreuzer and Schoeppe [3] was used during analysis of the IMP curves. The typical IMP curve (see Figure, A) is characterized by a short gradual, followed by a very steep,rise (2, 3). The increase of IMP begins 0.01-0.05 sec after the beginning of the ECG ventricular complex (see figure A, 1) and depends only slightly on the duration of the cardiac cycle. At point 3 (see figure, A), corresponding to the end of the period of isovolumic contraction, the rate of increase of the IMP falls sharply, after which some increase of pressure is observed in the wall of the left ventricle, up to a maximum, followed again by a slow, and then a fast decrease of the IMP. The interval between points 3 and 4 corresponds to the phase of expulsion of blood from the ventricle and that between points 4 and 5 to the period of relaxation of the myocardium in this part of the left ventricular wall (see figure, A).

Besides the typical form of IMP curve, in some cases bimodal curves are observed. On these curves, at the end of the isovolumic contraction phase, instead of a further, although slower, increase of pressure in the ventricle wall, a temporary decrease is observed, followed by an increase of the IMP to its maximum. This transient decrease of IMP is evidently due to an abrupt decrease in myocardial tension as a result of opening of the aortic valve and the rapid expulsion of blood from the ventricle. This suggestion is confirmed by the fact that the beginning of the fall on the IMP curve always coincides with the beginning of increase of aortic pressure (see figure, B). Evidently, the fall of IMP observed after the beginning of expulsion of blood from the ventricle is based on the same mechanism as the lowering of tension in the muscle with a rapid decrease in its length.

The onset of the increase of IMP in the basal and middle portions of the left ventricular wall usually precedes the onset of the increase of intraventricular pressure by 0.01-0.02 sec. In contrast to this, the increase of IMP in the apical portion either coincides with the beginning of increase of pressure in the ventricle or precedes it by not more than 0.01 sec. Since the end of the period of isovolumic contraction for all parts of the myocardium is determined by opening of the aortic valve, and mechanical activity in the middle and basal portions of the left ventricular wall begins sooner than in the apical region, the duration of the period of isometric myocardial contraction in the middle third and base of the left ventricular wall (0.068 sec) is longer than the corresponding index for the apical region of the heart (0.053 sec). Fundamentally similar, although less marked differences between the duration of the isovolumic period of the myocardium in different parts of the left ventricle were found by Osadjan and Randall [4].

Comparison of the IMP curve in different parts of the myocardium with the curve of intraarotic pressure shows that from a certain moment, despite the continued increase of IMP, the intraaortic pressure begins to fall. As a rule the intraaortic pressure reaches its maximum 0.03-0.09 sec before the peak of the IMP.

After closure of the aortic valve the intraventricular pressure falls rapidly to the diastolic level. In contrast the IMP usually falls much more slowly and reaches the diastolic level 0.04-0.06 sec after the intraventricular pressure.

Very clear differences were found between the IMP in different parts of the left ventricular wall. In the basal portion of the ventricle and in its middle third the IMP was close to the intraventricular pressure,

but in a few experiments it was below it. The ratio between the IMP and left intraventricular pressure (referred to later as the IMP/LV index) in the basal and middle portions varied from 0.66 to 1.4 with a mean value of 0.97. In 3 of 15 experiments the IMP/LV index was greater than 1, in 3 it was equal to 1, and in the other 9 it was less than 1. In contrast, in the apical region of the left ventricle the IMP/LV index was greater than 1 in all 9 experiments, ranging from 1.18 to 1.8. The mean value of this index for the apical region of the left ventricle was 1.38 ($P < 0.05$).

In all cases the IMP was higher in the deeper layers of the myocardium of the left ventrical wall than in the superficial layers.

One possible cause of the higher IMP in the apical region of the left ventricle is the smaller area of cross section of the wall in this part of the heart. Because of this, the tension of the myocardium in this portion of the ventricle must be higher than in portions with a significantly larger cross-sectional area of their wall, i.e., in the base and middle third of the left ventricle. Presumably the different architectonics of the myocardium in these regions, together with the unequal curvature of their wall, play an essential role in this phenomenon.

A characteristic feature is that the diastolic IMP is much higher than the intraventricular pressure. Whereas the final diastolic pressure within the left ventricle in normal conditions was 3.8 mm, the diastolic IMP was usually 10-15 mm, and it was always higher than the final diastolic pressure. Possible explanations of this are that during diastole, a certain proportion of the muscle fibers retain some of their tone, or that the blood flowing into the cavity of the ventricle stretches its walls, in which a certain level of tension develops because of the visco-elastic properties of the myocardium. Resolution of the radially acting force (intraventricular pressure) by the rule of the parallelogram of forces gives a tangential tension of the left ventricular wall significantly greater in magnitude [2].

The results described above demonstrate the functional heterogeneity of the myocardium of different portions of the left ventricle. It may be assumed that a further study of this problem will assist toward the understanding of variations in the pattern of hyperfunction of different portions of the myocardium in pathological states.

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